Streptococcus mutans Binding to Collagen and Fibrinogen in Nicotine

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Introduction: Our overall goal is to find the mechanism for atherosclerosis. Smokers have increased incidence of atherosclerosis. Atherosclerosis occurs when there is a build up of plaque in the arteries. There is evidence that *Streptococcus mutans* help cause this blockage. We have already proven that *S. mutans* produces more biofilm in certain concentrations of nicotine. Also, we have found that nicotine up-regulates *S. mutans* binding to proteins in certain concentrations; other labs have also demonstrated this. The intent of this study was to evaluate the binding of *S. mutans* to both collagen type I and fibrinogen, which are both proteins that are already present on the surface of endothelial cells lining arteries.
Methods: *S. mutans* UA159 was cultured in 0.00-4.00 mg/mL nicotine. The cells were killed in formaldehyde and then coated with biotin. The proteins studied were plated (1 ug/ml) on 96-well microtiter plates. In order to block the empty spaces that the protein did not bind to, 1% BSA in sodium bicarbonate buffer was added to the plate. Each nicotine dilution of *S. mutans* was added to the plate and the amount of binding was assessed. Extra-avidin HRP and OPD were added to the plate and the intensity was measured at an absorbance of 490 nm using a spectrophotometer.
Results: The intensity was directly related to the number of cells bound to the proteins. There was a significant increase in *S. mutans* binding when compared to the baseline for both collagen type I and fibrinogen. The binding was highest when *S. mutans* were cultured in 2 and 4 mg/mL nicotine.
Conclusions: The data collected suggests that collagen type I and fibrinogen contribute to the mechanism of atherosclerosis. When *S. mutans* are cultured in moderately high concentrations of nicotine, more binding of the bacteria to these proteins occurs.

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